

WHAT IS CLAIMED IS:

5 1. A transgenic mouse whose genome comprises a transgene encoding human small conductance calcium-activated potassium (SK) channel protein, splice variant B1 ("SK3-1B"), wherein the transgene is operably linked to a neuron-specific promoter, and wherein expression of the transgene results in ataxia.

10 2. The transgenic mouse of claim 1, wherein the expression of the transgene is limited to neurons.

15 3. The transgenic mouse of claim 1, wherein the mouse is fertile and transmits the SK3-1B transgene to its offspring.

4. The transgenic mouse of claim 1, wherein the SK3-1B transgene has been introduced into an ancestor of said mouse at an embryonic stage.

20 5. The transgenic mouse of claim 1, wherein the mouse is hemizygous for the human SK3-1B transgene.

25 6. The transgenic mouse of claim 1, wherein the mouse is homozygous for the human SK3-1B transgene.

7. The transgenic mouse of claim 1, wherein the mouse overexpresses SK3-1B in neurons relative to a control non-transgenic mouse.

30 8. The transgenic mouse of claim 1, wherein the promoter is a Thy1.2-SX promoter.

9. A transgenic mouse whose genome comprises a transgene encoding SK3-1B, wherein the transgene is operably linked to a neuron-specific promoter, and wherein expression of the transgene results in an intention tremor.

10. The transgenic mouse of claim 9, wherein the expression of the transgene is limited to neurons.

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11. The transgenic mouse of claim 9, wherein the mouse is fertile and transmits the SK3-1B transgene to its offspring.

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12. The transgenic mouse of claim 9, wherein the SK3-1B transgene has been introduced into an ancestor of said mouse at an embryonic stage.

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13. The transgenic mouse of claim 9, wherein the mouse is hemizygous for the human SK3-1B transgene.

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14. The transgenic mouse of claim 9, wherein the mouse is homozygous for the human SK3-1B transgene.

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15. The transgenic mouse of claim 9, wherein the mouse overexpresses SK3-1B in neurons relative to a control non-transgenic mouse.

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16. The transgenic mouse of claim 9, wherein the promoter is a Thy1.2-SX promoter.

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17. A transgenic mouse whose genome comprises a transgene encoding SK3-1B, wherein the transgene is operably linked to a neuron-specific promoter, and wherein expression of the transgene results in hyperexcitable behavior.

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18. The transgenic mouse of claim 17, wherein the expression of the SK3-1B  
transgene is limited to neurons.

5 19. The transgenic mouse of claim 17, wherein the mouse is fertile and transmits  
the SK3-1B transgene to its offspring.

10 20. The transgenic mouse of claim 17, wherein the SK3-1B transgene has been  
introduced into an ancestor of said mouse at an embryonic stage.

15 21. The transgenic mouse of claim 17, wherein the mouse is hemizygous for the  
human SK3-1B transgene.

22. The transgenic mouse of claim 17, wherein the mouse is homozygous for the  
human SK3-1B transgene.

20 23. The transgenic mouse of claim 17, wherein the mouse overexpresses SK3-1B  
in neurons relative to a control non-transgenic mouse.

25 24. The transgenic mouse of claim 17, wherein the promoter is a Thy1.2-SX  
promoter.

25 25. A method of screening biologically active agents that facilitate reduction of  
ataxia in vivo, the method comprising:

30 administering a candidate agent to a transgenic mouse according to claim 1, and  
determining the effect of said agent upon the level of ataxia.

26. A method of screening biologically active agents that facilitate reduction of  
intention tremors in vivo, the method comprising:

35 administering a candidate agent to a transgenic mouse according to claim 9, and

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determining the effect of said agent upon the level of intention tremors.

5       27.    A method of screening biologically active agents that facilitate improvement in hyperexcitable behavior, the method comprising:

administering a candidate agent to a transgenic mouse according to claim 17, and  
determining the effect of said agent upon hyperexcitable behavior.

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